Coronary heart disease: prevalence and dietary sugars in Scotland

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Abstract

Study objective – The aim was to investigate the effects of dietary intakes of different types of sugars (extrinsic, intrinsic, and lactose) and the dietary fat to sugar ratio on prevalent coronary heart disease (CHD).

Design – This was a baseline cross sectional survey of CHD risk factors.

Setting – Twenty two Scottish health districts were surveyed between 1984 and 1986.

Participants – A total of 10 359 men and women aged 40–59 years were screened as part of the Scottish Heart Health Study, and a further 1267 men and women aged 25–39 and 60–64 years were screened as part of the Scottish MONICA (monitoring trends and determinants in cardiovascular disease) Study. The response rates were 74% and 64% respectively.

Methods – Subjects completed a questionnaire which included sociodemographic, health, and food frequency information. Medical history, response to the Rose chest pain questionnaire, and results of a 12 lead ECG recording were used to categorise subjects into CHD diagnosed, previously CHD undiagnosed, or no CHD groups. The χ^2 statistic was used to determine whether the CHD groups differed in their sugar consumption, and multiple logistic regression analysis, with adjustment for other potential coronary risk factors, was used to calculate odds ratios for prevalent CHD by intake fifths of dietary sugars.

Main results – Men, but not women, differed in their sugar consumption by CHD group. The odds ratios showed a tendency for a U shaped relationship for extrinsic sugar intake with CHD prevalence, but no significant effect of the fat to sugar ratio (possible marker of obesity) on CHD was seen.

Conclusions – The results suggest that neither extrinsic sugar, intrinsic sugar, nor the fat to sugar ratio are significant independent predictors of prevalent CHD in the Scottish population, when the other major risk factors such as cigarette smoking, blood cholesterol concentration, and antioxidant vitamins intake are accounted for. These new data for different sugar types agree with the consensus view that total sugar intake is not a major marker of coronary heart disease.

Sugar intake per se has not been indicated in the aetiology of chronic disease other than dental caries. The cariogenicity of sugars has been attributed primarily to extrinsic sugar,¹ although meal frequency, cloyiness of food, and mouth retention time (which may be higher for complex carbohydrates) are of equal importance to cariogenicity, as is the relative intracellular or extracellular source of the sugars. Results from the cross sectional Scottish Heart Health Study suggested a possible lower prevalence of CHD in men with higher rather than lower total sugar intakes.² Since a low dietary fat to extrinsic sugar ratio has been related to a lower prevalence of overweight and obesity, independent of total energy intake, and there may be plausible energy related bases for this,³ it was considered pertinent to investigate further the relative roles of different sugar types to CHD prevalence in the Scottish population.

To investigate the true effect of dietary sugars on CHD prevalence, it is necessary to distinguish the dietary effects of having been diagnosed with heart disease from the initial dietary pattern which may give rise to the disease. In cross sectional studies this is best done by analysing those people with diagnosed CHD separately from those people who consider themselves healthy, but who on examination do have symptoms of CHD.⁴ In this report, multiple logistic regression analysis is used to examine the odds of having either diagnosed or previously undiagnosed CHD according to the amount of different sugars in the diet. Adjustment is made for the confounding factors such as relative weight, age, blood cholesterol, blood pressure, smoking, and dietary antioxidant vitamin intakes.

Subjects and methods

For the Scottish Heart Health Study, subjects were randomly selected in each of four age-sex groups from the lists of 10 general practitioners in each of 22 Scottish health districts to give a wide spread of CHD mortality. The selected patients were sent a letter, co-signed by their GP, with a questionnaire to complete and an invitation to attend a clinic for medical examination, as reported previously.⁵ A total of 10 359 men and women aged 40–59 years were recruited into the Scottish Heart Health Study (response rate 74%). Over the same period (1984–86) a further 1267 men and women aged 25–39 and 60–64 years were recruited using identical methods from two areas, Edinburgh and north Glasgow, as part of the international collaborative study of trends

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and determinants of cardiovascular disease (MONICA).⁶

Dietary information was obtained by food frequency questionnaire,⁷ CHD status by a medical history and the Rose chest pain questionnaire with additional evidence from a 12 lead ECG recording. The ECG, blood pressure, weight, and height were recorded at a clinic attendance where a non-fasted venous blood sample was also taken for various biochemical analyses, including blood cholesterol concentrations and serum cotinine as an independent marker of smoking status.⁸ Self reported smoking history was recorded using the WHO smoking questionnaire.⁹

Nutrient intakes were calculated from the food frequency questionnaire using standard portion sizes and the UK food composition tables in a computerised form. Validation studies in this Scottish population have been reported.^{10,11} Total dietary sugars were assigned to intrinsic, extrinsic, or milk sugar categories, based on Ministry of Agriculture, Food and Fisheries guidelines. Intrinsic sugar was that present naturally in whole fresh fruits, vegetables, and grains, plus 50% of the analysed sugars present in cooked, dried, or processed fruits. Extrinsic sugar (non-milk) was that sugar not naturally occurring within the intracellular structure of foods, plus 50% of the sugar in cooked, dried, or processed fruits. Lactose was sugar derived from milk, regardless of its location (that is, including lactose in chocolate, puddings, etc).

Study subjects were categorised into control, undiagnosed CHD, or diagnosed CHD groups² according to their response to the medical history and the Rose angina questionnaires,¹² and on the basis of the ECG recording. Controls were those without any evidence of CHD, and without a history of taking drugs for cardiovascular disease or hypertension. Undiagnosed CHD subjects were those who had no previous history of CHD, but who either responded positively to the angina questionnaire or exhibited the Q/QS pattern or ST-segment and T-wave changes on their ECG.¹³ The diagnosed CHD group were those who reported a previous medical diagnosis of myocardial infarction or angina. Subjects taking cardiovascular or hypertensive medication but with no other indication of CHD were omitted from further analysis.

STATISTICAL METHODS

Mean (SD) values are reported. Analysis of variance was performed on the appropriately transformed data to improve the normality of distributions: these were square root transformations for g/day intrinsic and extrinsic sugars; arcsine square root transformations for the % energy values of intrinsic, extrinsic, and lactose sugars; and logarithmic transformations for the fat to sugar ratios.

The χ^2 tests were used to assess the differences in distribution of CHD status by fifths of sugar intakes within sexes, and multiple logistic regression for determining odds ratios for the prevalence of CHD relative to the lowest fifth of intake for each sugar variable. Adjustment was made for age, total serum cholesterol, diastolic blood pressure, dietary antioxidant vitamins (vitamins C and E, and β -carotene), smoking, g/day of alcohol, weight, and height. Any additional effect of social class was investigated by including this as a covariate in one model, and total energy intake was also adjusted for when the odds ratios were determined on the g/day intake data.

Results

The response rates for the Scottish Heart Health and the MONICA studies were 74% and 64% respectively. Dietary data were obtained from an average of 95% of the total 5768 men and 5858 women who completed the studies.

Table 1 shows the number of men and women categorised into each of the CHD groups, and the mean dietary intakes of sugars and fat to sugar ratios in the different CHD groups. No significant differences were observed for women, while for men the percentage of energy from intrinsic sugar and lactose tended to be lower in the undiagnosed group, and the fat to sugar ratios were higher in both the diagnosed and undiagnosed groups than in the controls.

Differences between the proportion of diagnosed and undiagnosed people in each fifth of sugar intakes were found to be significant for men, with respect to extrinsic sugar (g/day and % energy), lactose (g/day), intrinsic sugar (% energy), and the fat to extrinsic sugar ratio, but not for women (results not shown). This meant that the undiagnosed and diagnosed groups

Table 1 Mean (SD) daily intake of sugars by coronary heart disease (CHD) group

	Men				Women		
	Control	Undiagnosed	Diagnosed	p	Control	Undiagnosed	Diagnosed
No	4340	772	411		4283	891	263
Intrinsic sugar:							
g/day	12.4 (7.7)	11.6 (7.2)	11.6 (7.2)	**	14.8 (7.7)	15.0 (8.0)	14.1 (7.9)
% energy	2.06 (1.40)	1.89 (1.32)	2.19 (1.51)	***	3.31 (1.92)	3.31 (1.97)	3.15 (1.81)
Extrinsic sugar:							
g/day	73.2 (45.0)	77.8 (51.2)	64.8 (49.1)	***	41.6 (30.8)	42.8 (32.2)	43.6 (35.9)
% energy	11.2 (5.4)	11.4 (6.1)	10.5 (6.1)	**	8.55 (5.1)	8.63 (5.39)	9.10 (5.96)
Lactose:							
g/day	20.6 (7.6)	20.5 (8.1)	19.6 (7.9)	*	19.1 (7.4)	19.0 (7.4)	18.3 (7.8)
% energy	3.34 (1.28)	3.25 (1.34)	3.53 (1.48)		4.14 (1.58)	4.05 (1.55)	4.02 (1.73)
Fat: total sugar:							
% energy	2.38 (1.16)	2.52 (1.51)	2.57 (1.41)	*	2.79 (1.30)	2.85 (1.34)	3.03 (2.54)
Fat: extrinsic sugar:							
% energy	4.28 (3.40)	4.69 (4.65)	5.10 (4.65)	**	6.45 (5.31)	6.72 (6.44)	4.31 (6.84)

Significant difference between male CHD groups by analysis of variance on appropriately transformed data (see statistical methods); * p < 0.05, ** p < 0.01, *** p < 0.001. No significant differences for women.

Table 2 Distribution of coronary heart disease (CHD) groups (%*) by intake fifths of different sugars

	Men (fifths)					<i>p</i>	Women (fifths)				
	1	2	3	4	5		1	2	3	4	5
Intrinsic sugar:											
Control	76	79	78	81	80		78	79	81	77	79
Undiagnosed CHD	16	15	14	12	13	*	17	17	14	18	16
Diagnosed CHD	7	6	8	7	9		6	4	5	5	5
Extrinsic sugar:											
Control	75	80	81	82	76		77	81	81	79	78
Undiagnosed CHD	15	13	13	12	16	***	18	15	16	17	16
Diagnosed CHD	11	7	6	6	8		5	5	4	4	6
Lactose:											
Control	77	79	82	80	77		76	79	79	79	80
Undiagnosed CHD	16	14	12	13	14	*	18	16	16	17	15
Diagnosed CHD	6	7	6	8	9		5	5	5	4	5
Fat: total sugar:											
Control	79	79	81	78	76		79	79	81	79	78
Undiagnosed CHD	15	13	13	15	15	*	16	15	18	16	17
Diagnosed CHD	7	8	6	7	9		5	4	5	4	6

* Percentage values were rounded to nearest whole number.

Significance level (χ^2) for men: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. No significant differences for women.

could be combined for women, but not for men. Table 2 shows the proportion of each CHD group in different fifths of sugar intakes. The χ^2 tests indicate that the distributions differed significantly between the diagnosed, undiagnosed, and control groups for men. For women no differences were observed, even when the two CHD groups were combined (data not shown).

The odds ratios (OR) for prevalent CHD by fifths of sugar intakes, adjusted for other potential coronary risk factors, are shown in tables 3 and 4. The diagnosed and undiagnosed groups are considered separately for men, but are pooled for women. For men (table 3), only two ORs are significantly different relative to the lowest fifth of intake (characterised by 95% confidence intervals (CI) which do not span 1). These are middle

fifths of extrinsic sugar intake, where the risk of diagnosed CHD is lower by 34% (CI 4,55) and the risk of undiagnosed CHD is lower by 28% (CI 5,46). Although the ORs do differ significantly between the fifths for undiagnosed CHD, there is no evidence of a dose-response effect for extrinsic sugar or for any of the other sugar variables examined.

The situation differs only slightly for women (table 4), in whom the risk of prevalent CHD (diagnosed or undiagnosed) is significantly lower in the middle fifth of intrinsic sugar intake, and shows a non-significant tendency to be lower in the middle fifths of extrinsic sugar intake. A slight, although again not significant, dose-response effect is seen for the ORs by fifths of the fat to sugar ratio. Significantly lower prevalent CHD occurs in the upper two fifths, with the maximum effect being a 24% (CI 2,41) reduction from the highest fat to sugar intake group compared with the lowest.

The results were unchanged when the sugar variables were expressed as a percentage of total energy intake rather than as g/day. Further adjustment for occupational social class had no significant effect on the results either.

Discussion

Similar analyses of such cross-sectional data have previously proved valuable for identifying the antioxidant vitamins as important dietary factors for predicting the prevalence of CHD in this Scottish population.⁴ Thus, the number of CHD cases in this analysis provides sufficient power to detect biologically significant effects of individual nutrients. However,

Table 3 Male odds ratios (OR) (95% confidence intervals (CI)) for prevalent diagnosed and undiagnosed coronary heart disease (CHD) by intake fifths of dietary sugars

	Fifths									
	1 OR	2 OR (CI)	3 OR (CI)	4 OR (CI)	5 OR (CI)	1 OR	2 OR (CI)	3 OR (CI)	4 OR (CI)	5 OR (CI)
Intrinsic sugar:										
Diagnosed CHD	1.0	0.75 (0.50,1.13)	1.07 (0.73,1.58)	0.92 (0.61,1.40)	0.98 (0.61,1.57)					
Undiagnosed CHD	1.0	1.00 (0.77,1.30)	1.02 (0.77,1.35)	0.85 (0.62,1.26)	1.18 (0.83,1.68)					
Extrinsic sugar:										
Diagnosed CHD	1.0	0.78 (0.55,1.11)	0.66 (0.45,0.96)	0.75 (0.51,1.10)	0.97 (0.66,1.41)					
Undiagnosed CHD	1.0	0.96 (0.71,1.23)	0.77 (0.58,1.02)	0.72 (0.54,0.95)	1.03 (0.78,1.37)*					
Lactose:										
Diagnosed CHD	1.0	0.99 (0.67,1.46)	0.84 (0.56,1.26)	1.05 (0.71,1.55)	1.20 (0.82,1.78)					
Undiagnosed CHD	1.0	0.97 (0.75,1.26)	0.83 (0.63,1.09)	0.95 (0.72,1.25)	1.22 (0.91,1.62)					
Fat: total sugars (% energy)										
Diagnosed CHD	1.0	1.23 (0.84,1.78)	0.84 (0.56,1.27)	0.74 (0.49,1.11)	1.01 (0.68,1.48)					
Undiagnosed CHD	1.0	0.79 (0.61,1.04)	0.84 (0.64,1.11)	0.98 (0.75,1.29)	0.92 (0.69,1.21)					

OR relative to the lowest intake fifth. Bold type identifies ORs which differ significantly from 1.0, and italic type identifies ORs which border on a significant difference from 1.0.

* Indicates a significant difference ($p < 0.05$) in the adjusted prevalences of CHD between intake fifths for that sugar variable.

Table 4 Odds ratios (OR) (95% confidence intervals (CI)) for prevalent coronary heart disease (CHD) (diagnosed and undiagnosed) in women by intake fifths of dietary sugars

	Fifths									
	1 OR	2 OR (CI)	3 OR (CI)	4 OR (CI)	5 OR (CI)	1 OR	2 OR (CI)	3 OR (CI)	4 OR (CI)	5 OR (CI)
Intrinsic sugar	1.0	0.90 (0.70,1.17)	0.72 (0.55,0.95)	1.06 (0.79,1.41)	0.91 (0.65,1.28)*					
Extrinsic sugar	1.0	0.86 (0.66,1.10)	0.82 (0.63,1.06)	0.94 (0.73,1.21)	1.17 (0.91,1.51)*					
Lactose	1.0	0.90 (0.71,1.16)	0.99 (0.78,1.27)	0.96 (0.75,1.23)	0.91 (0.70,1.17)					
Fat: total sugars (% energy)	1.0	0.81 (0.63,1.03)	0.87 (0.68,1.12)	0.77 (0.60,0.99)	0.76 (0.59,0.98)					

OR relative to the lowest intake fifth. Bold type identifies ORs which differ significantly from 1.0, and italic type identifies ORs which border on a significant difference from 1.0.

* Indicates a significant difference ($p < 0.05$) in the adjusted prevalences of CHD between intake fifths for that sugar variable.

these results do not suggest any significant role for the different dietary sugars in the prediction of CHD risk, since there is a noticeable lack of any dose-response effect. A dose-response effect is the best indicator of a true cause and effect relationship in the absence of any biologically plausible reason for a U shaped relationship (such as occurs for alcohol and CHD). While it is possible that the lowest and highest intake groups may be at higher risk of CHD as a result of other factors that have not been adequately adjusted for in the logistic regression models, there is no evidence to suggest that this is so.

These results seem to support the already large body of evidence which suggests that dietary intake of sugars has no specific bearing on CHD risk.¹ They extend the existing knowledge regarding sugar intake and CHD by clarifying that extrinsic sugar intake cannot be differentiated from the effects of intrinsic sugar or lactose. Furthermore, no coronary benefit seems to accrue from a relatively low fat and high sugar diet, which is associated with lower relative weight and lower prevalence of obesity. This seems to confirm the current view that obesity per se is not a major risk factor for CHD, and that the other risk factors, such as cigarette smoking and antioxidant vitamin intake (which were fully adjusted for in the analysis reported here), are more important predictors of CHD.

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